Some effects of nicotine on the central nervous system of chickens

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Effects of nicotine on behaviour and electrocortical activity of the fowl differed from those of muscarine. Intraventricular nicotine (0·5 μmol) had biphasic effects. Immediately after injection, the chicken squatted for 3–5 min, with no sign of muscle contracture. The eyes were usually closed and this stage was accompanied by slow frequency (3 to 5 potentials/s) large amplitude (400 μV) electrocortical potentials only distinguishable from those of sleep by superimposed irregular "spike" discharges also noted after intravenous injection (Key & Marley, 1962). Thereafter, the fowl stood with neck and wings extended, respiratory rate being increased to above 200/min; electrocortical activity returned to the pre-injection pattern. The effects on posture and respiration lasted 30–40 min. Body temperature was unaltered. Respiratory effects of nicotine (0·125 μmol) were potentiated by eserine (0·75 μmol). Intravenous pempidine (100 μmol/kg), but not similar doses of hyoscine, prevented behavioural, electrocortical and respiratory effects of nicotine (0·5 μmol). Intraventricular pempidine (1 μmol) elicited within 5 min the tachypnoea and late postural changes similar to those after nicotine; the electrocortical and early postural changes were not observed. When nicotine doses were spaced less than 40 min apart, the duration and intensity of its effects dwindled.

In fowls anaesthetized with chloralose, intraventricular nicotine (0·5 μmol) evoked apnoea for 15 to 40 s, after which respiratory amplitude briefly increased. Blood pressure usually rose by 25–70 mmHg, but sometimes a fall or a fall followed by a rise was observed. This contrasted with cats, in which intraventricular nicotine usually lowered blood pressure (Hall & Reit, 1966; Armitage & Hall, 1967a, b). Pressor effects of intraventricular nicotine lasted 1–2 min but were prolonged to 60–90 min after bilateral vagotomy and abolished by additional spinal cord division at C2.

Nicotine (0·125 μmol) micro-infused into the prosencephalon and particularly when given into the brain-stem, evoked behavioural and electrocortical effects like those immediately after intraventricular administration. Respiration and temperature were unaltered. When infused unilaterally into the telencephalon, the electrocortical slow wave and irregular "spike" discharge occurred ipsilaterally, a normal "sleep" pattern being observed on the contralateral hemisphere; the full behavioural changes developed, however. Pempidine (100 μmol/kg intravenously or intracerebrally 0·15 μmol), but not hyoscine in similar doses, prevented the effects of nicotine. Intracerebral pempidine had no agonistic effects.

Nicotine, like muscarine, has widespread responsive areas in the brain, although effective doses were much larger for nicotine. A short period of sleep-like behaviour and electrocortical activity was induced in contrast to arousal with muscarine. Although temperature was unaltered, the tachypnoea and posture with muscarine and nicotine were similar to those normally adopted for heat loss during hyperthermia.

A distinction between the effects of muscarine and nicotine on behaviour and electrocortical activity has not apparently been made hitherto.

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The effect of adrenaline on 5-hydroxyindole concentrations in the hypothalamus and thalamus of rat

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A subcutaneous injection of insulin (sufficient to induce seizures) increases the concentrations of 5-hydroxytryptamine (5-HT) and 5-hydroxyindole acetic acid (5-HIAA) in rat brain (Gordon & Meldrum, 1970) and inhibits 5-hydroxytryptophan decarboxylation in rabbit brain (Costa & Himwich, 1959).

Adrenaline is released from the adrenal medulla as a result of insulin-induced hypoglycaemia, and in the present experiments the effect of adrenaline on brain 5-HT metabolism has been observed.

Adrenaline (0·67 μg/g subcutaneously) was administered to male Porton strain rats of 140–160 g. After intervals ranging between 30 min and 24 h, during which food and water had been withdrawn, the animals were killed by decapitation. Blood glucose and concentration of 5-hydroxyindoles in the hypothalamus and thalamus were estimated as previously described (Gordon & Meldrum, unpublished). In control experiments, adrenaline was replaced by 0·5 ml normal saline.

There was a maximal increase of 5-HT (22%) and 5-HIAA (56%) 3 h after injection. 5-HT levels returned to normal at 4 h. 5-HIAA levels were still elevated

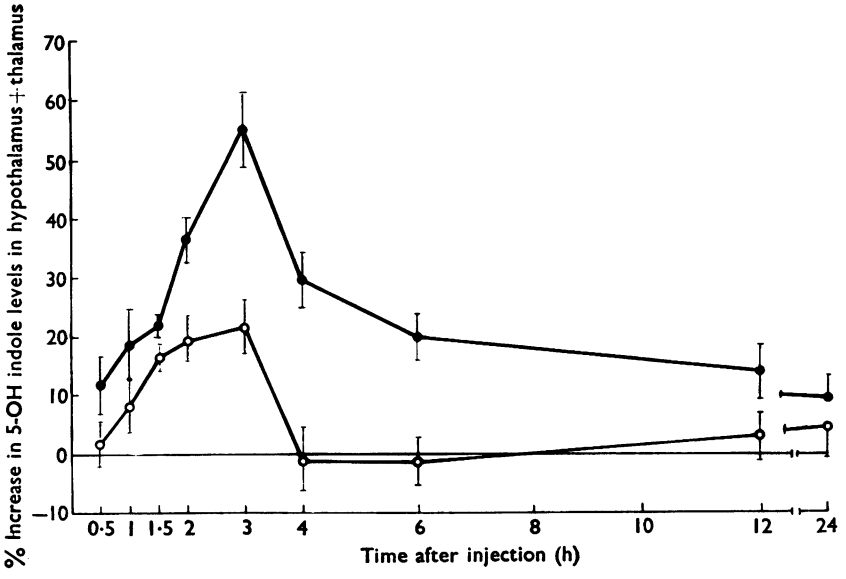

FIG. 1. Percentage increase (± % S.E.) in concentration of 5-HT (○–○) and 5-HIAA (●–●) in rat hypothalamus and thalamus after injection of 100 μg adrenaline. Number of experimental animals = 12; number of control animals = 12.